Table 1. BMD values and percentage of BMD change at the start of treatment with denosumab and during two years of withdrawal.

Previous Stop Dmab (n=83)			Stop Dmab (n=54)		Break Dmab1 (n=28)		Break Dmab2 (n=20)	
	(g/cm2)	T-score	(g/cm2)	T-score	(g/cm2)	T-score	(g/cm2)	T-score
DMO (mean)								
Lumbar spine	0,861 ± 0,1	-2,61	0,949 ± 0,1	-1,94	0,965 ± 0,2	-1,93	0,920 ± 0,2	-2,22
Femoral neck	0,735 ± 1	-1,96	0,774 ± 0,1	-1,71	0,758 ± 0,1	-1,84	0,740 ± 0,1	-2,02
Total femur	0,784 ± 0,1	-1,81	0,823 ± 0,1	-1,48	0,805 ± 0,1	-1,63	0,801 ± 0,1	-1,68
% change DMO								
Lumbar spine			12,2 ± 10,3		-5 ± 7,9		-5,44 ± 7,9	
Femoral neck			6 ± 8,5		-4,2 ± 3,9		-5,35 ± 5,8	
Total femur			3,9 ± 4,2		-5,2 ± 4,9		-0,33 ± 19,6	

OP0299

CLINICAL FEATURES ASSOCIATED WITH FRAGILITY FRACTURE AFTER DISCONTINUATION OF TREATMENT WITH DENOSUMAB: A CASE-CONTROL STUDY

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Background: Discontinuation of denosumab treatment causes an increase in bone resorption that has been linked to the emergence of vertebral fractures.

Objectives: To evaluate the association of clinical features and demographic characteristics with the emergence of new fragility fractures in patients with osteoporosis who interrupt treatment with denosumab.

Methods: Retrospective case-control study. Medical records of all patients with osteoporosis, from our local densitometry database, who received treatment with denosumab (at least one dose) and discontinue the treatment, have been studied.

Information was collected on demographic variables (age, sex), risk factors for osteoporosis (alcohol and tobacco consumption, personal history of fragility fracture and history of maternal hip fracture), secondary osteoporosis (due to early menopause, disease or osteopenizing treatment), previous treatment for osteoporosis, start and end date of denosumab treatment and subsequent treatments. All patients who suffered a fragility fracture from 6 months after the last dose of denosumab up to 20 months later were defined as a case. Prior treatment with bisphosphonate was considered to those who had received it for at least 1 year, and subsequent treatment with bisphosphonate was considered to those who received it immediately or up to 8 months after the last dose of denosumab.

Results: In total, 63 patients who discontinued treatment with denosumab were included. Ten of them presented fragility fractures (6 vertebral, 3 forearm and 1 hip). Two patients had a fracture after 24 months of the last dose of denosumab, so don't were considered cases. 61 patients were women (96.83%) and the mean age (SD) was 70.22 (8.95) years. The mean time (SD) of treatment with denosumab was 42.25 (17.32) months, with a mean (SD) doses of 7.85 (2.78). The reasons for interruption of denosumab were: improvement 39 (65%), dental procedure 9 (15%), patient decision 6 (10%), adverse event 4 (6.66%) and inefficacy 2 (3.33%). Demographic and clinical features by fragility fracture are shown in Table 1. We found that subsequent treatment with bisphosphonates after denosumab was initiated in less patients in the fracture group than in the control group (10% vs 42.9%; p=0.05), and we found no fractures in patients diagnosed with secondary osteoporosis. No statistically significant differences were found in the rest of the variables.

Table 1. Demographic and clinical features by fragility fracture.

	No fracture	Fracture	Total	p
Age, mean (SD)	70.2 (9.5)	70.5 (5.5)	70.2 (8.9)	0.956
BMI, mean (SD)	25.0 (4.8)	25.3 (3.2)	25.1 (4.6)	0.844
Secondary OP (%)	26 (49.1)	0 (0)	26 (41.3)	0.004
Tobacco (%)	17 (32.1)	2 (25.0)	19 (31.1)	0.687
Alcohol (%)	3 (5.7)	0 (0.0)	3 (4.9)	0.490
Previous fracture (%)	20 (37.7)	6 (66.7)	26 (41.9)	0.104
Mother hip (%)	6 (11.3)	0 (0.0)	6 (9.8)	0.316
Previous BP (%)	29 (67.4)	2 (50.0)	31 (66.0)	0.481
Denosumab dose, mean (SD)	7.9 (2.8)	7.5 (3.0)	7.9 (2.8)	0.676
Subsequent BP (%)	21 (42.9)	1 (10.0)	22 (37.3)	0.050

BMI: body mass index; BP: bisphosphonate; OP: osteoporosis; SD: standard deviation.

Conclusion: Sequential treatment with bisphosphonates and diagnosis of secondary osteoporosis are related to the absence of fractures after discontinuation of denosumab treatment.

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OP0300

A CROSS-SECTIONAL, MATCHED-PAIR ANALYSIS OF ACPA POSITIVE AND ACPA NEGATIVE RHEUMATOID ARTHRITIS PATIENTS COMPARING THE PREVALENCE OF OSTEOPOROSIS, FRAGILITY FRACTURES AND UNDERLYING RISK FACTORS

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Background: Rheumatoid arthritis (RA) is associated with increased systemic bone loss, leading to a high risk for fragility fractures. Especially anti-citrullinated protein antibody (ACPA) positivity is considered a risk factor for local bone erosions and systemic bone loss¹.

Objectives: The purpose of this study was to compare ACPA positive versus ACPA negative RA patients in terms of the prevalence of osteoporosis and fragility fractures and to identify differences in underlying risk factors that influence bone health.

Methods: Rh-GIOP is an ongoing prospective observational study collecting and analyzing disease- and bone-related data from patients with chronic rheumatic diseases or psoriasis treated with glucocorticoids (GC). In this cross-sectional analysis, we performed a matched-pair analysis, matching 114 ACPA positive to 114 ACPA negative RA patients according to age (5-year-steps), sex, and body mass index (BMI, 2-unit-steps). Descriptive analyses were performed, with values displayed as mean ± standard deviation for continuous variables. Non-parametric tests were used at a two-sided significance level of 5% to compare differences in underlying and potential risk factors without adjustment for multiple testing.

Results: At same mean age (63.9 ± 10.2 years) and BMI (27.9 ± 5.6 kg/m²), the matched groups had a female proportion of 82.5%. ACPA positive patients had a significantly longer mean disease duration (13.9 vs 9.9 years, p<0.001), a higher mean cumulative GC-dose (22.3 vs 13.2g, p<0.01) and mean duration of GC therapy (10.1 vs 6.6 years, p<0.01). There was no significant difference in the prevalence of osteoporosis as defined by dual-energy X-ray absorptiometry (DXA) (18.4 vs 20.2%), nor in the prevalence of vertebral (7.0 vs 5.3%) or non-vertebral fractures (31.6 vs 29.8%). C-reactive protein levels as a marker of disease activity were significantly higher in ACPA positive patients (mean: 8.8 vs 4.3 mg/l, p=0.02), while mean disease activity score (DAS)28 levels were slightly lower in ACPA positive patients (2.4 vs 2.7, p=0.05). No difference in health assessment questionnaire (HAQ) score was found. RA-specific treatments were similar, especially concerning current mean daily GC-dose (6.7 vs 4.9 mg/day), except for Rituximab and targeted synthetic disease modifying anti-rheumatic drugs (DMARDs) which were more commonly used in ACPA positive patients (9.6 vs 2.6%, p=0.05) and (5.3 vs 0%, p=0.029), respectively. ACPA positive patients did not differ significantly from ACPA negative patients in specific anti-osteoporotic treatment, nor in the prevalence of comorbidities or concomitant medication. There were no significant differences in bone-specific laboratory parameters.

Conclusion: In a cross-sectional analysis of our cohort, the prevalence of osteoporosis and fragility fractures was similar between ACPA positive and ACPA negative RA patients, despite longer disease duration and GC-treatment in ACPA positive patients. This is remarkable since it implies that ACPA negative patients are at a similar risk for osteoporosis and associated fractures. Optimal management of disease activity with or without GCs may represent a mainstay in preventing disease-related comorbidities such as osteoporosis.

References: [1]Steffen, U., Schett, G., & Bozec, A. (2019). How Autoantibodies Regulate Osteoclast Induced Bone Loss in Rheumatoid Arthritis. *Frontiers in Immunology*, 10, 1483. doi:10.3389/fimmu.2019.01483

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